STUDY OF NEUROLOGICAL SOFT SIGNS AND PSYCHOPATHOLOGY IN CHILDREN OF PATIENTS WITH SCHIZOPHRENIA AND AFFECTIVE DISORDER

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INTRODUCTION

Since the 1950s, prospective studies of individuals at heightened risk for future serious psychopathological conditions have been considered a potentially valuable strategy for studying the origins and development of such conditions. The high-risk strategy is based on the fact that offspring born to parents with psychosis are at increased risk for the later development of psychosis and other forms of psychiatric illness.

The risk for a child for developing schizophrenia is 13% if one parent is affected and 40% if both parents have schizophrenia. In children of bipolar parent there is 25% chance that the child will have a mood disorder and 50-75% if both parents have a mood disorder compared to the rates of 1% in the general population for both major mental disorders. Because the morbid risk rate of schizophrenia and affective disorder in the general populations is low to select subjects at random for prospective evaluation, subjects chosen based on empirically elevated risk, allows investigation to assess the morbid risk rates to the offspring and to discern environmental factors associated with the development of such disorders in those who are predisposed.

Childhood neurobehavioral deficits in offspring of parents with psychosis can be predictors of psychosis in adulthood. Early HR studies pointed to a wide prevalence of psychopathology among young relatives at increased genetic risk. Recent studies suggest that young HR relatives have neurobehavioral deficits and structural, physiological, and neurochemical brain abnormalities that may date back to childhood or earlier. Prospective studies of young relatives at risk for schizophrenia and affective disorder can also shed light on premorbid precursors.

Neurological deficits have been included in descriptions of schizophrenia since Kraepelin first defined dementia precox. Studies have reported a greater than normal overall number of neurological signs in patients with adult and childhood schizophrenia and in the offspring of schizophrenia (Reider and Nichols, 1979). In fact, the presence of neurological signs is so strong a finding that a congenital neurointegrative deficit is believed to be at the base of at least some subtypes of the illness.

Behavioral disorders and neurological soft signs are all possible manifestations of the genetic predisposition to psychotic disorders. Several studies have suggested a link between adult schizophrenia and certain behavioral or neurological signs in childhood, and it is now well established

that early signs of disorder can be found during infancy and childhood. However, these signs are not exclusive to schizophrenic illness, although they occur with a greater prevalence in this group.

Mirsky et al (1995) in their 25 year follow up of children at genetic risk for schizophrenia found that children who eventually developed schizophrenia spectrum disorders were identifiable by cognitive-psychophysiological, neurointegrative, and social/personality traits in the preteenage period. As Hanson et al., reported in 1976, which abnormalities best define the schizophrenia vulnerable group among the offspring of schizophrenics, and when they first appear are questions that are not yet conclusively answered.

Neurological abnormalities could represent an important early biological marker for schizophrenia risk (Walker 1994), as a higher rate of neurological abnormalities early in life has been found among individuals who later develop schizophrenia (Fish 1987; Hans et al., 1999). However for neurological abnormalities to function optimally as a biological marker for schizophrenia risk, we need to know the specific form of neurological abnormality related to schizophrenia risk, its distribution among high-risk and normal-risk individuals, its personal stability over different ages, and its specificity for risk for schizophrenia versus other psychoses. Debate continues about which specific neurobehavioral signs show the greatest sensitivity and

specificity to schizophrenia, and whether specific or general deficits are better indicators of vulnerability to schizophrenia.

As there are few comparative studies which have investigated the specific neurobehavioral deficits that differentiate the children of parents with schizophrenia from those with a parent suffering from affective disorder, this study aimed at determining the neurobehavioral signs that are specific to children of schizophrenic parent as against those with a parent suffering from Affective disorder.

REVIEW OF LITERATURE

Historical background of Neurological soft signs

Bender in 1956 introduced the concept of 'Soft Neurological Signs' (Synonym: Minor neurological dysfunction) which are defined as non normative performance on a variety of motor and / or sensory tasks by people not mentally retarded and without focal neurological signs. As an index for cognitive and behavioral dysfunction NSS have been studied by many worker (Hertzig et al 1996, Quitkin et al 1976, Kolakowska et al 1985). Most child neurologists and psychiatrists view them as subtle developmental immaturities (Shapiro et al 1979, Lunswing et al 1992). They are labeled "Soft" because they run a developmental course, by which it is usually meant that they diminish in prevalence and severity with age and do not have any clear locus of origin. Such signs are not pointers to disease in the traditional sense and their pathological roots are obscure. Most, however, can be elicited readily and reliably (Rutter et al, 1970). A different group of signs are considered "Soft" because they are minor in degree and because they are difficult to detect in a reliable fashion. Such signs would include reflex or tone asymmetries.

Soft signs occur in many otherwise normal children. Thus Adams et al. (1974) found that 10 per cent of 9-11 year-olds without behavior or learning problems had NSS. Rutter et al (1970) had reported that 14 per cent of normal 10-11-year olds showed mirror movements. Shaffer et al (1978) concluded that disturbed children with neurological dysfunction may develop specific psychiatric syndromes in later life. It is not clear, whether later disorder is directly related to persisting C.N.S. abnormalities or whether the association is more oblique.

Hertzig (1981), who studied a neurologically deviant population, found that although there was a diminution in amplitude and range of signs found in an individual child, children with a sign at one age are likely to show signs, not necessarily the same ones 5 years later. Shaffer et al (1985) also found that a larger proportion of children who had soft signs at age 7 continue to show such signs at age 17.

It has been suggested that Neurological soft signs reflect a failure in integration within or between sensory and motor systems (Griffiths et al., 1998) while others advocate deficits at subcortical level (basal ganglia, brainstem or limbic system) (Kennard, 1960; Mosher et al, 1971). It is possible that soft signs reflect an impairment of the normal cortico-cortical and cortico-subcortical interneuronal anatomical connections, which have

been proposed as one of the fundamental pathophysiological substrates of schizophrenia (Friston and Frith, 1995).

Imaging studies on the anatomical correlates of NSS suggest that it might represent a clinical sign of a perturbed cortical-subcortical connectivity (Dazzan et al, 2004).

Significance of Neurological soft signs in Schizophrenia

Neurological 'soft signs' are commonly found in individuals with schizophrenia (Gupta et al, 1995) and appear to have a developmental origin, having been identified in studies of high-risk children (Erlenmeyer-Kimling & Cornblatt, 1987), birth cohorts (Jones et al, 1994), prodromal illness (McGorry et al, 1995), patients with negative symptoms (Malla et al, 1997), and drug-naive patients (Schroder et al, 1991, Venkatasubramaniam et al., 2003).

The relationship between neurological soft signs and the genes for schizophrenia is uncertain. Though it is well established in schizophrenic patients, its etiopathological and clinical significance continues to be unclear (Browne et al., 2000). It is unclear whether these abnormalities are directly related to processes that result in the symptoms that mark the advent of psychosis or are simply epiphenomenal indices of a generally disturbed brain development. It is also unclear whether neurological abnormalities related to

schizophrenia are associated primarily with risk for schizophrenia or even affective psychosis.

Heinrich and Buchanan in their review in 1988 concluded that the uncertainty of their meaning in schizophrenia reflects not the unreality of the finding but limitations in our knowledge and there certainly is no basis for dismissing neurological signs as in principle nonspecific or soft. Abnormal signs in neurology have traditionally been assumed to be highly informative about the nature and location of disease once the significance of these signs is understood. There is no reason to assume that this is different in the case of schizophrenia.

Neurological soft signs are consistently reported to be more frequent in patients with schizophrenia than in healthy controls (Ismail et al, 1998; McNeil et al, 2000). It was showed that schizophrenia patients are more differentially characterized by neurological abnormalities representing "hard signs," while siblings of schizophrenia patients are more characterized by "soft signs" (Ismail et al.,1998). Nichols and Chen (1976), found greater concordance for signs among the monozygotic twins. This finding, coupled with observations on the ratio of concordance between full siblings and first cousins, is compatible with a genetic origin of soft signs. They reflect brain dysfunction in most cases and are influenced by heredity.

Venkatasubramaniam et al., (2003) reported higher neurological signs in never treated patients and lack of association with illness duration and suggested neurodevelopmental etiopathogenesis.

In schizophrenia impairments are found in three higher-order functional areas: the integration of more complex sensory units, the coordination of motor activity, and the sequencing of motor patterns. While one might be tempted to postulate that these areas reflect abnormalities in the parietal lobe, the cerebellum, and the frontal lobe, respectively, this would be an unwarranted and undoubtedly erroneous simplification. Indeed, several of the authors suggest that the problem is likely to be subcortical, either in the basal ganglia and brainstem or in the limbic system (Mosher et al., 1971).

High resolution MRI studies show that higher rates of soft neurological signs (both motor and sensory) were associated with a reduction of grey matter volume of subcortical structures (putamen, globus pallidus and thalamus). Signs of sensory integration deficits were additional associated with volume reduction in the cerebral cortex, including the precentral, superior and middle temporal and lingual gyri. Dazzan et al., 2003 concluded that neurological soft signs are associated with regional grey matter volume changes and that they may represent a clinical sign of the perturbed cortical-subcortical connectivity that putatively underlines psychotic disorders.

Patients with neurological soft signs demonstrated significantly poorer performance on neuropsychological tasks that assessed timed motor speed and motor coordination (e.g., finger tapping, the Purdue Pegboard task, and part B of the Trail Making Test). These findings continued to be significant even after lifetime medication exposure, extrapyramidal symptoms, and abnormal involuntary movements were used as covariates. These findings support the notion that soft signs are a manifestation of a localizable behavioral deficit of the systems that are involved in motor speed, coordination, and sequencing and are not indicative of global cognitive impairment. The specific deficit in motor abilities is consistent with the types of neurological soft signs that are most frequently reported and suggests involvement of frontal/subcortical circuitry in schizophrenia (Flashman et al., 1996).

Comparative studies of NSS in Schizophrenia and Affective disorder

Manschreck & Ames (1984) had reported neurological soft signs in 92% of schizophrenic patients, 52% of affective disorder and 5% of control subjects. Similarly Krebs et al., (2000) reported higher incidence of total score of NSS in schizophrenic patients than mood disorder. Boks et al., (2004) reported that a particular set of NSS shows specificity for Schizophrenia. Other studies found no difference between schizophrenia & bipolar (Nasrallah

1983) and concluded that the two are indistinguishable in terms of neurodysfunction (Whitty et al 2006).

Neurological soft signs children of parents with Schizophrenia

Fish (1980) described her experience of serial neurological assessments beginning in infancy of the offspring of schizophrenic and nonpsychotic mothers. While she did not find a stable neurological defect, she reported a pattern of erratic neurointegrative development over time. This maturational dysfunction, termed as "pan-developmental" retardation, was significantly more frequent in the offspring of schizophrenic mothers and was associated with serious psychopathology, including psychosis, later in life.

Marcus et al., in an Israeli high-risk study (1974, 1985) reported that children who were younger than 11 years of age and had a schizophrenic parent had more neurological abnormalities than matched control subjects. The difference reached significance in a "median split analysis" that compared the more deviant half of each group. This analysis was employed because only a portion of the offspring would be expected to have the vulnerability to the illness. Impairments were prominent in fine motor performance, right-left orientation, motor coordination and overflow, and sensory integration. They

found a similar excess of neurological dysfunction in the offspring of a Danish sample.

The Israel study of children of schizophrenics found that 44% of the offspring of schizophrenics had signs of neurointegrative deficits (Marcus et al, 1985). The Jerusalem infant development study (1999) found that a disproportionate number of offspring of schizophrenic parent (42%) and especially male offspring (73%) showed poor neurobehavioral functioning relative to offspring of non- schizophrenic parent.

Individuals at genetic risk for schizophrenia may display lifelong neurobehavioral signs that are indicators of vulnerability to schizophrenia and that are associated with psychiatric disturbance in general and schizophrenic spectrum disorders specifically.

All offspring who received schizophrenia spectrum diagnoses by adolescence showed a pattern of poor neurobehavioral functioning across developmental periods. Children with schizophrenic parents, when compared with children with healthy parent or parent having other psychiatric disorders, were more likely to show neurobehavioral dysfunctioning in perceptual–cognitive and motoric areas. A stable subgroup (40%) of the offspring of schizophrenics showed dysfunctioning during infancy and school age. None of the offspring of nonschizophrenic parent showed dysfunction during both age

periods. Perceptual – cognitive signs were strongly linked to parental diagnosis and infant dysfunctioning. Motoric signs, but not cognitive signs, were related to pregnancy and birth complications. These findings provide further support to the schizotaxia hypothesis that some neurointegrative deficits may reflect vulnerability to schizophrenia and that these deficits are clearly apparent at school age, long before the onset of illness (Marcus et al., 1993).

Virtually all of the children identified as showing poor neurobehavioral functioning had both perceptual-cognitive and motoric signs. Parental diagnosis of schizophrenia was more closely linked to the perceptual-cognitive component than to the motor component. Which suggest that the perceptual-cognitive signs are better indicators of schizophrenic diathesis than motoric signs.

Erlenmeyer Kimling and Cornblatt (1987), made the observation that both motoric signs and attentional problems were increased in children at risk for schizophrenia, but that only attention was closely related to actual schizophrenic breakdown in their sample.

Lawrie et al (2001) reported that sensory integration abnormalities were more frequent in high-risk subjects than in healthy controls. They concluded

that the lack of associations with psychotic symptoms and genetic liability to schizophrenia suggests that soft signs are non-specific markers of developmental deviance that are not mediated by the gene(s) for schizophrenia.

The male offspring of schizophrenic were much more likely than female offspring to be represented in the children with poor neurobehavioral functioning at school age. This is consistent with other evidence of poorer premorbid history in male schizophrenics than female schizophrenics and even with the suggestion that male and female patients may manifest different subtypes of schizophrenic illness (Goldstein and Tsuang, 1990).

Among the offspring of schizophrenics who were followed up from infancy through middle childhood, 40% of the offspring of schizophrenics showed poor neurobehavioral functioning during both periods. The Swedish High-Risk Study, found no personal stability of neurological abnormalities between the neonatal period and 6 years of age ((Mc Neil et al.,1993, Blennow et al., 1991). The Jerusalem Infant Development Study (Hans et al., 1999; Marcus et al., 1987) showed modest stability between infancy and adolescence (14–21 years of age) but strong stability between school age (8–13 years) and adolescence. The total score for neurological abnormalities at 22 years was

significantly positively correlated with the total score at 6 years of age for the total high-risk group, the offspring of mothers with schizophrenia and the normal-risk offspring but not for the offspring of mothers with affective psychosis.

Those signs observed in children not at risk for schizophrenia may be related to other nonschizophrenic disorders such as retardation, learning disabilities, and attention deficit disorder. It appears that most of the poorly functioning offspring of nonschizophrenics show change and improvement over time. Such a pattern of behavioral change is likely due to normal plasticity in early brain development that may not be available to genetically vulnerable children.

Finding suggests that genetic risk for schizophrenia may make the fetal brain more vulnerable to mild perinatal insults that have no effect on infants who are not at genetic risk. In the JIDS study, children genetically at high risk but with no perinatal birth complications showed neurobehavioral signs, and children genetically at low risk with perinatal birth complications showed no neurobehavioral signs. Based on these it was concluded that genetic vulnerability alone is sufficient for expression of neurobehavioral signs, particularly perceptual cognitive signs, but genetic vulnerability coupled with perinatal insults may produce greater motor deficits.

Psychopathology in PreSchizophrenic individuals

Nearly century ago, Eugen Bleuler described a variety of nonpsychotic abnormalities present before the onset of schizophrenia. Other report have observed in individual patients somatic complaints, obsessions and compulsive behaviors, anxiety, panic, depressive symptoms, and other psychopathologic features preceding the onset of schizophrenic episodes. Schizophrenia often may be a result of long-term interactions between vulnerability and stress. In such case, the process leading to full schizophrenia might be detected early by patterns of prodromal abnormalities.

Quantitative determination of an antecedent psychopathologic condition could help advance understanding of these processes (Tien et al., 1992). Follow-back studies – examinations of the childhood histories taken from those who are unaware of the schizophrenic outcome-have quite consistently supported a view that certain patterns of childhood behavior were consistently found in patients with schizophrenia. Two major patterns, antisocial behavior and asocial (schizoid, withdraw) behavior, have been found. Bower et al., (1960) in an investigation of the high school behavior of male preschizophrenics through teacher interviews, found that about 50% had been withdrawn, 20% delinquent, and the other 30% had miscellaneous or no marked problems. A review of child guidance records of adult schizophrenics

by Nameche et al (1964) also found both withdrawn and acting-out types of childhood behavior disorders. Watt et al, (1998) found that preschizophrenic boys were often disruptive in the later grades of school, and 52% of them they categorized as "unsocialized – aggressive". Watt's group found that the two patterns described previously were sex-related, boys being more antisocial and girls more socially withdrawn. About 20% of the antisocial boys seen at the child guidance center later received a diagnosis of schizophrenia.

Though shyness and withdrawal were not shown to be highly associated with schizophrenia in these follow-up studies, it should be noted that in many studies the factors that best predict chronicity of schizophrenia have been found to be social isolation and poor sexual adjustment (Nameche et al., 1964).

Psychopathology in children of parents with Schizophrenia

Reider et al., (1979) concluded that behavioral disorders of the hyperkinetic-antisocial or the withdrawn asocial type, and neurological soft signs, are all possible manifestations of the genetic predisposition to schizophrenia. Menkes et al., (1967) followed up 14 hyperactive children who had been seen at child psychiatry out patient clinic and found four of them to be psychotic. Mednick and Schulsinger (1968) related classroom disturbance to later breakdown.

Data suggest that deterioration in social and intellectual functioning between childhood and adolescence is associated with the development of a negative symptom syndrome in schizophrenia. The premorbid deterioration appears to be an early prodrome of the disorder (Kelly 1992).

The Copenhagen High-Risk Study found that a significant aggregation of schizophrenia (16.02%) and other nonaffective, nonorganic psychosis (4.6%), and cluster A personality disorders (21.3%) occurred among the offspring of schizophrenic mothers compared with the controls (1.9%, 0.9% and 5%, respectively). No evidence of increased aggregation of (psychotic and non-psychotic) affective disorders was noted among the offspring of schizophrenics.

Mirsky (1988) reported that poor attentional skills (on a digit cancellation task) in the preteen to early teenage years (average age 11) were highly correlated with spectrum disorders in adulthood.

Children at risk for schizophrenia are so at risk for interpersonal behavior problems during middle childhood, particularly social withdrawal (Hans et al. 1992).

Hanson et al., (1976) proposed that a combination of poor motor skills, test variability, and behavior abnormalities may identify predisposed

individuals. Impaired visual-motor coordination and greater distractibility (Lifshitz et al., 1985); lower sociometric rankings by their peers (Sohlberg and Yaniv 1985); and impaired interpersonal relations, work and play activities, self-esteem, and mood (Nagler and Gluect 1985) were among the other areas of dysfunction reported.

Comparative studies of high-risk children

Erlenmeyer-Kimling et al., (2000) assessed the predictive relationships between neurobehavioral variables examined in mid-childhood and later schizophrenia-related psychoses in offspring of schizophrenic, affectively ill, and normal parents in the New York High-Risk Project. Offspring were tested with neurobehavioral measures at 7–12 years of age and assessed in mid-adulthood for axis I diagnoses. For offspring of schizophrenic parents, childhood deficits in verbal memory, gross motor skills, and attention identified 83%, 75%, and 58%, respectively, of the subjects with schizophrenia-related psychoses; 50% were identified by all three variables combined.

False positive rates in subjects who did not develop schizophrenia-related psychoses ranged from 18% for those with deficits in attention during childhood to 28% for those with deficits in memory. The three variables had low deficit rates in the offspring of the other two parental groups and were not

associated with other psychiatric disorders in any group. They concluded that schizophrenia-related psychoses in adulthood are distinguished in subjects at risk for schizophrenia by childhood deficits in verbal memory, gross motor skills, and attention.

The findings suggest that deficits in these variables are relatively specific to schizophrenia risk and may be indicators of the genetic liability to schizophrenia. Low sensitivity and false positive rates for prediction of schizophrenia-related psychoses in the offspring of affectively ill and psychiatrically normal parents suggest that deficits in each mediating variable representing neurobehavioral performance are comparatively unique to risk for schizophrenia.

The total prevalence of impairment in the offspring of schizophrenic parents compared with offspring of affectively ill parents was 24% versus 0%, respectively, for attention, 37% versus 12% for memory, and 34% versus 9% for motor skills. Sensitivity, in correctly predicting schizophrenia-related psychoses, was unusually high for verbal memory (83%) and gross motor skills (75%).

The nonpsychotic offspring of schizophrenic parents who were among the 10% falsely classified when all three variables were combined are of interest because they appear to be carriers of some of the susceptibility genes

for schizophrenia and may yield information about other factors that are needed for development of the overt illness.

In a longitudinal study conducted by Schubert et al**.,** (2005) with a 93% rate of effective follow-up, the authors investigated neuropsychological impairment and its relation to neurological abnormality at a mean age of 22.3 years in offspring with heightened risk for schizophrenia and affective psychosis, and normal-risk offspring.

Offspring with genetically heightened risk for schizophrenia showed significantly impaired verbal memory, selective attention, and grammatical reasoning, compared with normal-risk offspring. Having impaired verbal memory, attention, and grammatical reasoning functions identified a significantly larger subgroup (16%) among offspring with heightened risk for schizophrenia than among offspring with heightened risk for affective psychosis (0%) and among normal-risk offspring (3%).

Multiple neuropsychological functions were significantly related to neurological abnormality in offspring with heightened risk for schizophrenia and in normal-risk offspring but not among offspring with heightened risk for affective psychosis. They concluded that the neurocognitive dysfunction attending heightened risk for schizophrenia is likely based on genetically

mediated neurodevelopmental factors, with schizophrenia and affective psychosis belonging to different biological spheres.

Children of person with schizophrenia are more impaired on attention test than the children of nonschizophrenic, major psychiatric disorders (Erlenmeyer-Kilmling 1985; Rutschmann et al. 1986). This research has suggested that impaired attention in children at risk for schizophrenia may represent a biobehavioral marker for the disorder.

Unlike the result obtained with the attention/distraction test, in which the poorer scores were obtained by those children who would later be diagnosed as having schizophrenia spectrum disorders, the least responsive subjects at age 11 were the probands who would later be diagnosed as having affective spectrum disorders. As indices of vulnerability, the attentional and arousal measures are predictive of differing types of psychopathological development.

Earlier investigations have shown that approximately one-third of schizophrenic patients exhibited obvious premorbid behavioral abnormalities. Prospective and retrospective cohort studies have found differences in childhood social and intellectual functioning between preschizophrenic children and the general population but have not found similar significant

effects for children destined to develop affective psychosis. However, it has been suggested that poor premorbid functioning is a predictor of vulnerability to psychosis among patients with major depressive disorder.

One interesting finding is that positive self-esteem variable may be a protective factor in subjects at risk for development of a psychiatric disorder. Higher IQ scores serve to some extent as a protective factor in the development of an affective disorder rather than a schizophrenia spectrum disorder (Nathan et al. 1993).

Offspring of schizophrenics receiving schizophrenia spectrum diagnoses by adolescence showed a pattern of poor neurobehavioral functioning across developmental periods. Individuals at genetic risk for schizophrenia may display lifelong neurobehavioral signs that are associated with psychiatric adjustment generally and schizophrenic spectrum disorder specifically (Hans et al 1999).

Although risk is elevated for the biological offspring of schizophrenic and affective disorder parents most children of such parents will never develop schizophrenia. This underscores the need to refine our ability to identify those individuals within at risk group who are at highest risk for the disorder.

OBJECTIVE

1. The objective of this study is to assess the neurological soft signs and psychopathology in children of schizophrenia and affective disorder patients.

2. To find the association between Neurological soft signs and psychopathology.

3. To determine the neurological soft signs and psychopathology that are specific to children of schizophrenia patients as compared to those of affective disorder patients.

MATERIALS AND METHODS

Setting

The study was conducted at Government Rajaji Hospital, Madurai which is a teaching hospital with tertiary care facility. The project protocol received the approval of the ethics committee of the institution.

Period of study

9 months from November 2005 – July 2006.

Study design

The study was a cross-sectional observational study. The study population included the offsprings of the parents attending General Psychiatric out-patient department. The children were recruited based on the following selection criteria.

Inclusion criteria

1. School going children in the age group 5-15 years.

2. Children with a parent diagnosed as schizophrenia or affective disorder as defined by ICD-10.

Exclusion criteria

1. H/o general medical on neurological illness in child or parent.
2. Children of parents diagnosed as Schizoaffective disorder or Severe Depression with psychosis
3. Children living away from either of the parent.
4. Presence of psychiatric disorder in the other parent.
5. Parents and children who were not cooperative for the study.

Patients were recruited using separate random charts for the two diagnostic groups. 60 children belonging to 46 families with an affected parent (Schizophrenia- 22, Affective disorder- 24) were included. There were 12 families with 2 siblings (Schizophrenia-8, Affective disorder- 4) fulfilling the above criteria and 1 family (Affective disorder) with 3 siblings. Thus a total of 152 individuals (60 children + 92 parents) participated in the study.

While screening patients it was noted that most of the hebephrenic remained unmarried or were separated. There were only 7 children whose parents had a diagnosis of catatonic schizophrenia, the other 23 children in the

schizophrenic group had their parent diagnosed as paranoid-schizophrenia. In the affective disorder group 17 children had a bipolar parent and 13 were unipolar offsprings.

Assessment and diagnosis

Following the receipt of written consent from the mentally healthy parent after disclosing the nature of the procedure, the parents were interviewed and diagnosed according to ICD-10. The diagnosis of the affected parent was ascertained after discussing with the assistant professor. The unaffected parent was screened for psychiatric illness using Brief Psychiatric Rating Scale.

The assessment of the unaffected parent and child was done by the primary investigator while the assessment and recruitment of the affected parent was done by a second investigator. The primary investigator was blind to the diagnosis of the affected parent.

Tools employed

1. A **semi structured proforma** compiled for recording the socio demographic variables, birth, development, medical history and scholastic performance of the child and details about the parental illness; which includes age of onset, illness duration, treatment response and burden on family. Burden on family was quantified by

assessing- financial burden, disruption of routine family activities, disruption of family interaction, disruption of family leisure, impact on physical and mental health of family member.

2. **ICD-10:** International Classification of mental and behavioral disorders was employed to define the diagnosis of the parent and the child.

3. **Brief Psychiatric Rating Scale** was used to screen the healthy parent. It includes 18 items rated on a 7 point scale (0-not present, 7-most severe).

4. **Revised neurological examination for subtle signs** by Martha Bridge Denckla (1985) which is a revised version of the Physical and Neurological Examination for Soft Signs (PANESS) was used to evaluate the children for neurological soft signs. It involves a timed series of sensory motor coordination tasks in addition to evaluation of posture and gait. It allows assessment of laterality of function in addition to quantification of motor impersistence and postural maintenance. Performance on the PANESS was significantly correlated with WISC-R indices sensitive to brain dysfunction, and behavioral factors implicated in the description of minor neurological dysfunction (Holden 1982). It includes a section for physical examination and another for assessment of NSS. One or both sections can be employed.

The section that contained the neurological examination for soft signs was alone used for this study.

5. **Child Behavior Check List** (CBCL: 4-18) by Achenbach (1991) was used to assess the behavior problems in the children. It includes 113 items and contains the total problem scale, two broadband dimensions – Internalizing problems and externalizing problems and eight cross – informant syndromes – Aggressive behavior, Delinquent behavior, somatic complaints, Anxious / Depressed, Attention problems, Social problems and Withdrawn behavior (Vaughn et al, 1997).

The internalizing problems scale is comprised of the anxious/ depressed somatic complaints and withdrawn subscales. The externalizing problems scale is composed of the aggressive behavior and delinquent behavior subscales. Scores on the scales of the CBCL are reported in the form of raw scores as it was recommended that raw scores on CBCL behavior syndromes and problems scales be used in research (Achenbach 1991). Past research conducted utilizing the CBCL has demonstrated its validity and reliability in clinical settings (Shekim et al., 1986, Bird et al., 1987, Biederman et al., 1993).

The parent and child were interviewed together and separately. The instruments mentioned above were used for assessment. The scales were

applied to parents and children as applicable. Illiterate parents were assisted to fill in the CBCL.

After establishing adequate rapport and after the child fully understood what is expected of him or her, the neurological evaluation was done. Proper instruction and clear demonstration were given in an identical manner to all the children and a positive atmosphere was maintained throughout the neurological evaluation for subtle signs (Denckla 1985).

ANALYSIS

The statistical analysis was done using Chi-square test and t- test.

RESULTS

Socio Demographic Variables of The Children of parents of the two diagnosis groups were analyzed using chi- square no significant difference was made out (Table1).

Table I

Demographic variables of the Children

S.No.	Variables		Diagnosis of parent		χ^2 test
			SCHIZ	AFF.D	
1	Age Group	<10	13	15	χ^2= 0.6027
		>10	17	15	p > 0.05
2	Gender	Male	16	18	χ^2=0.6027
		Female	14	12	p > 0.05
3	Domicile	Rural	11	12	χ^2=0.6027
		Urban	19	18	p > 0.05

Table 2

Birth and Developmental Variables

S.No.	Variables		Diagnosis of parent		χ^2 test
			SCHIZ	AFF.D	
1	Consanguinity	Present Absent	11 19	12 18	χ^2= 0.6027 P > 0.05
2	Birth Order	1 >1	14 16	18 12	χ^2=0.6027 P > 0.05
3	Birth complications	Present Absent	4 26	2 28	χ^2=0.6027 P > 0.05
4	Speech Delay	Present Absent	2 28	1 29	χ^2=0 P > 0.05

The birth and developmental variables of the children were also found to be comparable using chi-square test (Table II). Birth complications encountered were Low birth weight in 3 children of schizophrenic parent, Neonatal complication in 1; the 2 children in affective disorder group had

Neonatal complication. On evaluation of the developmental milestones it was found that speech was delayed in 3 of the children. There was no delay in motor milestones in any of the children.

Table 3

Scholastic Performance of children

Variables		**Diagnosis of parent**		**χ^2 test**
		SCHIZ	**AFF.D**	
School Performance	Poor	11	12	$\chi^2 = 0.2670$
	Good	19	18	$p>0.05$

The scholastic performance of the children was assessed based on parental reports; there was no statistical difference between the two groups using chi-square test.

Table 4

Illness variables of affected parent

S.No.	Variables		Diagnosis of parent		χ^2 test
			SCHIZ	AFF.D	
1	Gender	Male Female	12 18	10 20	χ^2= 0.0670 p > 0.05
2	Age of onset	≤25 years >25	15 15	11 19	χ^2=0.6109 p > 0.05
3	Duration of illness	≤2 years >2	8 22	12 18	χ^2=1.6968 p > 0.05
4	Family history	Positive Negative	13 17	6 24	χ^2=2.5381 p > 0.05
5	Premorbid personality	Introvert Extravert	15 15	4 26	**χ^2=7.7021** **p<0.01***

The illness variable of the affected parent in the two groups was analyzed using chi-square test. The two groups did not differ significantly in

gender distribution, age of onset, chronicity of illness or family history. But significant difference was found in premorbid personality. Larger number of affective disorder patient had premorbid extravert personality than schizophrenic patients. (Table 4)

Table 5

Treatment Response and Burden on Family

S.No	Variables		Diagnosis		χ^2test & p value
			SCHIZ	AFF.D	
1	Treatment Response	Satisfactory	25	30	$\chi^2 = 2.4685$
		Unsatisfactory	5	0	p>0.05
2	Burden On family	High	22	17	$\chi^2 = 11.4172$
		Low	8	23	**p<0.001***

Burden on the family due to mental illness was assessed by evaluating the effect of illness on the various aspects of family functioning including financial burden, disruption of routine family activities, disruption of family leisure, disruption of family interaction, impact on physical and mental health of family members. A positive score in more than 3 areas of impairment out of 6 of the above was taken to imply greater burden on family based on report of the healthy parent. It was found that the burden was significantly high in

families having a schizophrenic individual though there was no significant difference in treatment response (Table 5).

Table 6

Comparison of Demographic Birth & Developmental Variable with NSS Scores

S. No.	Variables		Total NSS			t test
			N	Mean	S.D	
1.	Age group	< 10 yrs	28	24.0395	9.2986	**0.0156**
		> 10 yrs	32	17.6875	9.2123	
2.	Gender	Male	34	22.3235	9.1344	2.5725
		Female	26	18.7307	10.4050	
3.	Domicile	Rural	36	21.75	9.7845	0.9519
		Urban	24	19.2917	9.8090	
4.	Consanguinity	Present	23	14.7391	8.8842	0.6617
		Absent	37	21.4054	10.3748	
5	Birth Complications	Present	6	25	11.6790	0.9517
		Absent	54	20.2963	9.5653	
6	Developmental	Present	3	26	3.6056	**2.2363***

	Delay- Speech	Absent	57	20.4912	9.9447	
7	Scholastic Performance	Poor Good	23 37	22.6087 19.6216	9.7316 9.7763	0.8874

***p < 0.05 significant**

The demographic variables and the birth and developmental variables of the child were analyzed. It was found that all variables except speech delay did not affect the total score of Neurological soft signs (Table 6). Children who had history of delay in speech development scored significantly high.

Table 7 shows that the gender of the parent and age of onset significantly influenced the scores on PANESS. Scores were significantly high for children of female parent and those with early age of onset. Family history and treatment response did not influence the scores.

Table 7

Comparison of Illness variables & Total Scores of NSS

S. No.	Variables		Total NSS			t test
			n	Mean	S.D	
1.	Gender of parent	Male	22	17.3182	9.3472	**2.1538***
		Female	38	22.7631	9.5900	
2.	Age of onset	≤25 years	26	26.1539	8.6781	**4.1327****
		> 25 years	34	16.6470	9.0248	
3.	Family history	Positive	19	23.0526	9.7268	1.2383
		Negative	41	19.7073	9.7500	
4.	Treatment response	Satisfactory	55	20.8727	9.8188	0.2619
		Unsatisfactory	5	19.6	10.4547	

*** p<0.05 significant**

**** p<0.001 significant**

Table 7a

comparison of premorbid personality and PANESS scores in the two groups

Diagnosis	Premorbid personality	N	Mean	S.D.	t test
SCHIZ	Introvert	15	25.93333	7.9952	1.8391
	Extrovert	15	20.13333	9.2340	
AFF.D	Introvert	4	21	14.7648	0.3785
	Extrovert	26	18.1154	9.6305	

p >0.05 not significant

It was found that parental premorbid status did not significantly influence the neurological performance of the child in the two diagnostic groups (Table 7a).

Table 8

Comparison of NSS of children of Parents of the two Diagnostic Groups

Diagnosis	N	Mean	S.D.
Schizophrenia	30	21.1	9.4553
Affective Disorder	30	18.6129	10.2577

t = 0.6230

p = 0.5357 Not significant

The total scores of Neurological soft signs on the PANESS scale-Revised Neurological examination for subtle signs were high for all children. There was no statistically significant difference between children of schizophrenia patients and those of Affective disorder patients on applying t-test (Table 8).

Table 9

Comparison of CBCL scores of Children of parents of two diagnostic groups

Diagnosis	n	Mean	S.D.
Schizophrenia	30	4.4333	5.8586
Affective Disorder	30	3.4667	3.9369

t = 1.0783

p = 0.2854 – Not Significant

On comparing the scores on the child behavior checklist it was found that there was no significant difference between the children of schizophrenic parent and mood disorder parent (Table 10). The average score of the children were low in both diagnostic groups.

Table 10

Comparison of NSS & CBCL scores

NSS / CBCL	**<18**	**18-24**	**>24**	**n**
0	4	6	8	18
1-3	9	6	2	17
4-6	5	6	7	18
7-9	1	0	0	1
>10	1	1	4	6
n	20	19	21	60

On analyzing the CBCL and the PANESS scores it was found that there was no correlation between that two scores and that both were in dependent variables. (Table 10)

Table 11

Comparison of NSS & CBCL scores of catatonic vs paranoid schizophrenia

Diagnosis		NSS Scores			CBCL Scores			
	n	<18	18-24	24	0	1-3	4-6	>10
Catatonia	7	1	0	6	3	1	1	2
Paranoid	23	6	10	7	8	5	6	4

p> 0.05 Not Significant

Table 11 a.

Scales	Diagnosis	Mean	S.D.
PANESS	Catatonia	21.1428	8.5133
	Paranoid	21.7826	8.9237
CBCL	Catatonia	6	7.5498
	Paranoid	4.4783	6.3380

p>0.05 not significant

On comparing the scores of children of parent having paranoid schizophrenia with those suffering from catatonic schizophrenia no significant difference was found using t test. (Table 11 & 11a)

Table 12

Comparison of NSS & CBCL score in children of Bipolar & Unipolar

Diagnosis		NSS Scores			CBCL Scores			
	n	<18	18-24	24	0	1-3	4-6	>7
Bipolar	17	8	4	5	3	6	8	0
Unipolar	13	6	3	4	4	5	3	1

p> 0.05 Not significant

Table 12 (a)

Scales	Diagnosis	Mean	S.D.
PANESS	Bipolar	18.4118	10.4407
	Unipolar	18.6154	10.2351
CBCL	Bipolar	3.2941	2.2849
	Unipolar	2.7692	2.7127

$p > 0.05$ Not significant

Table 12 shows the comparison of scores of total Neurological soft signs and the scores on child behavior check list between the children of bipolar parents and those of parents suffering form unipolar affective disorder. No statistical difference was made out between the two subgroups (table 12a).

Tables 13

Qualitative analysis of Abnormalities in subscales of PANESS in the two groups

Subjects	**Schizophrenia (%)**	**Affective disorder (%)**
Gait	90	70
Dysrhythmia	60	80
Impersistence	53	30
Involuntary movt	6	3
Repetition	100	93
Sequencing	93	86
Overflow-Excess	80	60
Overflow Total	83	67
Overflow L>R	37	27

The prevalence of abnormality on individual subscales of the PANESS was compared between the two groups in a qualitative manner. (Table 13)

There was no significant gender difference in the two diagnostic groups (table 14 & 15). On analyzing the association between gender of parent and of the child on the scores of PANESS it was found that female children of male parent with affective disorder had significantly low scores.

Table 14

Gender Distribution in the Schizophrenic Group

Parent / Child	Male	Female	χ^2
Male	8	8	0.3516
Female	4	10	

$p > 0.05$ Not Significant

Table 14a

Gender Distribution in the Affective disorder Group

Parent / Child	Male	Female	χ^2
Male	6	12	1.2054
Female	4	8	

p> 0.05 Not Significant

Table 15

Comparison of Gender of parent and Child on the PANESS Scores

Parent		Child				
		Sex	**N**	**Mean**	**S.D**	**t**
SCHIZ	**Male**	Male	8	20.375	10.267	-0.2734
		Female	8	21.5	3.873	
	Female	Male	4	26	6.7823	0.6136
		Female	10	23.4	11.0474	
AFF.D	**Male**	Male	6	18.1667	7.9	**3.2460***
		Female	12	5.75	4.113	
	Female	Male	4	23.25	10.1096	0.5808
		Female	8	18	9.1496	

*** p <0.01 significant**

Table 16

Comparison of scores of children with ICD-10 diagnosis with the normally functioning children

Child Diagnosis	n	Mean	S.D	t
Present	4	19.5	14.7083	0.1818
Absent	56	20.8571	19.5297	

p >0.05 not significant

On analyzing the association between ICD – 10 diagnosis of child on the total scores on PANESS no significant difference was found between these children the normally functioning children.

DISCUSSION

Comparison of the two diagnostic groups found no statistical difference in the socidemographic variables, birth, development and scholastic performance of the children of parents belonging to the two diagnostic groups. The illness variables of the parent- age of onset, duration of illness, family history (other than affected parent) were also found comparable on applying chi-square test. The parents however differed on their premorbid personality. Only 13% (4/30) of the parent with affective disorder had premorbid introvert personality as against 50% (15/30) of schizophrenic parent.

It was also found that burden on the family was significantly high for families of schizophrenic patient as compared to those of affective disorder,

though treatment response was reported satisfactory in both the diagnostic group.

On analyzing the association between the variables of the children and the affected parent on the total scores on PANESS it was found that sociodemographic variables did not significantly affect the total scores on PANESS. Ismail et al., (1998) had also reported no association between neurological abnormalities and sociodemographic characteristics.

The children of younger age group did not score significantly high as compared to older children, though NSS are considered developmental and a decrease in scores with age was expected. The results point to an indirect evidence that there could be persistence of atleast some of the NSS in these children as evidenced by earlier studies (Shaffer et al., 1985). However follow up is required to confirm these findings in these children.

Consanguinity and birth complications did not significantly affect the total scores on PANESS. The Jerusalem infant development study (1999) had concluded that genetic vulnerability coupled with perinatal insults may produce greater motor deficits. Though the scores were high in the children with birth complications it did not reach statistical significance. This

ascertains the genetic nature of NSS underscoring the attribution of early central nervous system insults in the causation of such abnormalities.

The study found that children who had developmental delay – particularly speech, had more neurological soft signs as compared to children who showed normal development. The British Birth Cohort study (1946) reported that children of schizophrenics had three times the number of speech problems as controls and had found that speech problems emerged as a significant risk factor for schizophrenia.

On analyzing the effect of sex of the affected parent and that of child in the two diagnostic groups, it was found that children of female parent had significantly higher neurological soft signs as compared to children of male parent. The study found that the female children of male parent with affective disorder scored significantly low. This variation in scores as influenced by gender of parent and child could probably reflect the differential inheritance.

The scores were also significantly high for children of parents with early age of onset of illness. This needs further investigation as it can provide clue about the underlying pathophysiology resulting in early breakdown. However other illness variables which are prognostic indicators of parent's illness like family history of mental illness other than the parent, premorbid

personality or treatment response of the affected parent did not significantly influence the scores.

The average scores on child behavior check list were low in both the groups. Although Shah et al., had reported higher scores in children of schizophrenic parent the scores were low which could be attributed to the fact that the scale was not translated into the local language. Studies have also reported that parent's report of children is underrated as compared to self report. Though some studies reported higher rates of pychopathology in children of bipolar parent (Carlson et al., 1993), Wals et al., (2001) concluded that it was not high.

There was no significant difference in the total scores of NSS in the children of parents of the two diagnostic groups. There was also no significant difference on the mean total score on CBCL.

There was no association between the scores of neurological soft signs and psychopathology. The two were found to be independent variables.

There was also no significant difference in the scores of the children of paranoid and catatonic schizophrenia or between bipolar and unipolar affective disorder subgroups.

Qualitative analysis of individual subscales found that relatively higher percentage of children of schizophrenic parent had abnormalities as compared to those of affective disorder parent in repetitive movements (100% vs 93%), sequencing (93% vs 86%), overflow-excess for age (83% vs 67%), overflow asymmetry – left > right (37% vs 27%).

Although earlier studies have quoted higher rates of psychopathology in children of psychotic parents, it was found that only 4 children had fulfilled ICD-10 diagnosis; 1-Attention deficit and hyperactivity disorder, 1-Enuresis, 1-Encopresis and 1 child fulfilled the criteria for hebephrenic schizophrenia. It was noted that these children especially the child diagnosed as Schizophrenic did not score high on PANESS. Lawrie et al., (2001) concluded that the lack of associations with psychotic symptoms and genetic liability to schizophrenia suggests that soft signs are non-specific markers of developmental deviance that are not mediated by the gene(s) for schizophrenia. They are rather trait like deficits.

Analyzing the children who scored high on both scales it was found that children with PANESS score > 24 and CBCL > 10 belonged to the schizophrenic group. ¾ were male children. It was noted that all 4 children with deviant scores were children of schizophrenic parent (n = 4/30, 13%). It is to be noted that only 13% of the children of schizophrenic parent are at risk

of developing the disorder. Follow up of these children can throw light on whether these children are the ones who are at highest risk for developing schizophrenia. Studies of the high-risk offspring of parents with schizophrenia at different ages have detected a subgroup with an especially high frequency of neurological abnormalities and with a higher risk for later development of schizophrenia-related psychopathology (Asarnow 1988; Marcus et al.,1985). The size of the subgroup (25%–50%) has varied across studies, possibly because of differences in methods and definitions. Earlier studies had also reported that male offspring were overrepresented in the poorly functioning group (Jerusalem infant development study, 1999).

The type, frequency, personal stability, and specificity of neurological abnormalities for offspring of women with different psychotic disorders were investigated in the Swedish High-Risk Study. It was reported that compared with the normal-risk offspring and the offspring of mothers with affective psychosis, this high-scoring subgroup among the offspring of mothers with schizophrenia contained notably more subjects which corroborates with the results of this study.

Schubert and Mc Neil (2004) reported that the adult offspring of mothers with schizophrenia had significantly more neurological abnormalities which is against the findings of this study. They also found that neurological

abnormalities at 22 years were significantly associated with neurological abnormalities at age 6 among the total high-risk group. They concluded that high levels of neurological abnormalities are found in a substantial proportion of offspring of mothers with schizophrenia but not offspring of mothers with affective psychosis and suggested that familial risk for schizophrenia is associated with neurodevelopmental disturbance that is manifest throughout life and belongs to a different biological continuum from that of affective psychosis.

Studies have reported that patients with affective disorders and their relatives generally show more neurological abnormalities than healthy comparison subjects but lower rates of neurological abnormalities than patients with schizophrenia and their relatives (Blennow et al., 1991)., but results vary across both high-risk and prepatient groups (Cannon et al., 2002;Kimling E L and Cornblatt B 1987). This question is related to unresolved issues concerning whether schizophrenia and affective psychosis belong to the same biological continuum and what the definitional limits for each of these disorders are somatically (Cardno et al.,2002).

The presence of neurobehavioral signs in the affective disorder group is a reminder that the measures used in this and many other studies involve many aspects of brain function and are not exclusive to schizophrenic illness even

though they occur with a greater prevalence in individuals at risk for schizophrenia.

LIMITATIONS

1. The major limitation of the study is its small sample size which limits the interpretation of the findings.

2. It was a cross sectional study. As soft signs are considered developmental, prospective follow up of the children exhibiting neurological soft signs would give better understanding about its correlation with vulnerability to psychotic illness.

3. Parents rating of their children's behavioral abnormalities could have resulted in possible underrating. It would have been better if supplemented by children's self report or teacher's rating.
4. The scales were not standardized for Indian children.

5. Finally, as in all high-risk studies, the ultimate interpretation of the data will depend on information about the adult psychiatrics status of the individuals being studied which needs follow up of these children.

CONCLUSION

As identification of the population at risk is the first step to prevention, major progress could be achieved with the help of such simple cost-effective clinical tools. Though there are no specific neurobehavioral signs to identify risk for individual illness it was found that most children exhibited neurological impairment higher than expected for age. Psychoeducating the parents about the possible risk for children and serial follow up can help to identify the children in the prodromal phase of illness before significant impairment results.

Prospective studies conducted with large samples of well-assessed parents and children is needed in order to further our understanding of the nongenetic factors that contribute to the development of mental disorders in

children of psychotic parents. Such information is necessary for the development of programs aimed at preventing, or at least attenuating symptom development in these children.

Rates of separation and divorce among persons suffering from schizophrenia and affective disorder are higher than in the general population. Consequently, some children of these parents are exposed to a "double risk," namely, heredity and detrimental psychosocial influences, while others are exposed to only the latter risk. Little is known, however, about the influence of this double risk on the development of mental disorders among the offspring of such parents during childhood and adolescence.

Currently, however, there is no gold standard tool to identify which of these children have inherited vulnerability for mental disorder. While some of these children are at increased risk because of an inherited vulnerability, all of them are exposed to detrimental psychosocial influences because they are being raised by a parent with a severe and chronic mental disorder which needs further investigation.

Clinical implications

- Children of psychotic parents are at higher risk for neuro-behavioural deficits in childhood and adolescence.

- Prospective longitudinal studies tracking the development of these children are needed in order to identify the determinants of mental disorders in this high-risk population.
- Psychiatric evaluations of a patient with schizophrenia and affective disorders must be extended to the entire family in order to allow for early intervention of symptomatic children.

RECOMMENDATIONS

In order to determine whether there is a persistence of neurobehavioral impairment from childhood to adulthood, large samples of children need to be followed through the periods of risk for the major mental disorders with assessments of their neurological and psychosocial functioning at various ages in order to understand more fully the precursors of the adult disorders.

In addition, the prevalence rates of disorders among these children need to be documented by age and by gender. A more precise understanding may result when gender and age are considered together. Since children do not

often develop major mental disorders before puberty, reports of results before and after puberty would be most useful.

The influence of the gender of the parent on the risk of mental disorders in the children also needs to be clarified. Association between the prevalence of mental disorders in the children and both the age of onset, severity and chronicity of the parent's disorder needs further investigation.

Studying anatomical correlates of soft signs in children at risk can give better understanding of the clinical usefulness of the scale as a screening tool.

APPENDIX - 2

GENERAL PROFORMA- CHILD

S.No:

Name:

Age: **Sex**: M / F

Address:

Rural/Urban Religion:

Language: Tamil/Others

Name of Father: Name of Mother:

Consanguinity: + / -- Deg: 2 / 3 / 4 Family tree:

Family Type: Nuclear / Extended / Joint / Disrupted

Affected Parent: Father / Mother **Hospital No.**

BIRTH HISTORY:

1. Mother's condition during pregnancy: ill / well : Details about illness-
2. Drug consumed if any: No / Yes-
3. Delivery: FT / PT Hosp / Home N / CS / Instrumental-Indication:
4. Birth weight- ____ LBW / Normal / Not known
5. Neonatal complications: Asphyxia / Seizures / Jaundice / Others

DEVELOPMENTAL MILESTONES:

1. Motor Development:Head control :Normal/ Delayed, Sitting Normal/Delayed
   Standing: Normal/ Delayed Walking: Normal/ Delayed
2. Speech Development: Normal/ Delayed
3. Toilet training- Normal / Delayed Enuresis: + / --

MEDICAL HISTORY

1. Injuries 2. Seizures 3. Meningitis / Encephalitis
4. Any major physical illness 5. Any physical Anomalies
6. Mental illness in the past

SCHOOL: Class: Academic: Good / Average / Poor H/O failure-
School refusal / Learning disability

DETAILS ABOUT UNAFFECTED PARENT

Name: **Age:** **Sex:** M / F

Education: Primary / Secondary / Hr. Sec / Graduate

Occupation: Unskilled / Skilled / Clerical / Professional Income:

Past H/O Mental illness: Yes / No

Family H/O MI / MR / Suicide / Substance / Seizure / PD

BPRS:

DETAILS ABOUT AFFECTED PARENT

S.No **Hosp.No**

Name: **Age:** **Sex:** M / F

Education: Primary / Secondary / Hr. Sec / Graduate

Occupation: Unskilled / Skilled / Clerical / Professional Income:

Past H/O Mental illness: Yes / No

Family H/O MI / MR / Suicide / Substance / Seizure / PD

PREMORBID TRAITS:

SUBSTANCE ABUSE: Nicotine / Alcohol / Cannabis / Others: Abuse / Dependence

OTHER MEDICAL ILLNESS:

PSYCHIATRIC HISTORY

Duration of illness

Precipitating factors: Life event

Onset: Acute / Insidious

Course: Continuous / Episodic- No. of Episodes-

No. of admissions in the past:

Nature of Rx: Drugs / ECT / Psychotherapy / Rehabilitation

Compliance to Rx: Regular / Irregular

Response to Rx: Satisfactory / Unsatisfactory

Effect of illness on Family

1. Financial Burden – Present/ Absent
2. Disruption of Routine family activities – Present/ Absent
3. Disruption of Family Leisure – Present/ Absent
4. Disruption of family Interaction – Present/ Absent
5. Physical Health of others – Affected / Unaffected
6. Mental Health of Others – Affected / Unaffected

DIAGNOSIS –ICD:

APPENDIX - 3

THE BRIEF PSYCHIATRIC RATING SCALE

0 – Not assessed 1 – Not Present 2 – Very Mild 3 – Mild
4 – Moderate 5 – Moderately Severe 6 –Severe 7–Extremely Severe

1. Somatic Concern
2. Anxiety
3. Emotional Withdrawal
4. Conceptual Disorganization
5. Guilt Feelings
6. Tension

7. Mannerisms and Posturing
8. Grandiosity
9. Depressive Mood
10. Hostility
11. Suspiciousness
12. Hallucinatory Behavior
13. Motor Retardation
14. Uncooperativeness
15. Unusual Thought Content
16. Blunted Affect
17. Excitement
18. Disorientation

TOTAL SCORE:

APPENDIX - 4

CHILD BEHAVIOR CHECK LIST FOR AGES 6-18

CHILD'S FULL NAME TODAY'S DATE:

CHILD'S GENDER Boy / Girl AGE: BIRTHDATE:

CHILD'S ETHNIC GROUP OR RACE

GRADE IN SCHOOL _____NOT ATTENDING SCHOOL

PARENTS' USUAL TYPE OF WORK, even if not working now. (Please be specific — for example, auto mechanic, high school teacher, homemaker, laborer, lathe operator, shoe salesman, army sergeant.)

FATHER'S TYPE OF WORK

MOTHER'S TYPE OF WORK

THIS FORM FILLED OUT BY:

Your gender: Male / Female

Your relation to the child: Biological Parent / Step Parent / Grandparent Adoptive Parent / Foster Parent / Other (specify)

Please fill out this form to reflect your view of the child's behavior even if other people might not agree. Feel free to print additional comments beside each item and in the space provided. Be sure to answer all items.

I. Please list the sports your child most likes Compared to others of the same age, about how much time does he/she spend in each one? For example: swimming, baseball, skating, skate boarding, bike, riding, fishing, etc. How well does he/she take part in each?

None

a. _____

b. _____

c. _____

II. Please list your child's favorite hobbies, activities, and games, other than sports, For example: stamps, dolls, books, piano, crafts, cars, computers, singing, etc. (Do not include listening to radio or TV.) Compared to others of the age about how much time he/she spend in each? How well does he/she do each one?

None

a. _____

b. _____

c. _____

III. Please list any organizations, clubs, teams, or groups your child belongs to. Compared to others of the same age, how active is he/she in each?

None

a. _____

b. _____

c. _____

IV. Please list any jobs or chores your child has For example: paper route, babysitting, making bed, working in store, etc. (Include both paid and

unpaid jobs and chores.). Compared to others of the same age, how well does he/she carry them out?

None

a. _____

b. _____

c. _____

V. 1. About how many close friends does your child have? (Do not include brothers & sisters)

None 1 2 or 3 4 or more

2. About how many times a week does your child do things with any friends outside of regular school hours? (Do not include brothers & sisters)

Less than 1, 1 or 2 3 or more

VI. Compared to others of his/her age, how well does your child:

Worse Average Better

a. Get along with his/her brothers & sisters? Has no brother or sister

b. Get along with other kids?

c. Behave with his/her parents?

d. Play and work alone?

VII. 1.Performance in academic subjects. Does not attend school because

_____

Check a box for each subject that child takes

a. Reading, English, or Language Arts

b. History or Social Studies

c. Arithmetic or Math

d. Science

e. _____

Other academic subjects–for example: computer courses, foreign language, business. Do not include gym, shop, driver's ed., or other non academic subjects.

Failing / Below Average/ Average/ Above Average

2. Does your child receive special education or remedial services or attend a special class or special school? No/Yes —kind of services, class, or school:

3. Has your child repeated any grades? No Yes—grades and reasons:

4. Has your child had any academic or other problems in school? No /Yes—please describe:

When did these problems start? _____

Have these problems ended? No Yes–when?

Does your child have any illness or disability (either physical or mental)? No Yes—please describe:

What concerns you most about your child?

Please describe the best things about your child.

Below is a list of items that describe children and youths. For each item that describes your child now or within the past 6 months, please circle,

0 = Not True (as far as you know) 1 = Somewhat or Sometimes True 2 = Very True or Often True

0 1 2 1. Acts too young for his/her age

0 1 2 2.Drinks alcohol without parents' approval (describe):_____

0 1 2 3. Argues a lot

0 1 2 4. Fails to finish things he/she starts

0 1 2 5. There is very little he/she enjoys

0 1 2 6. Bowel movements outside toilet

0 1 2 7. Bragging, boasting

0 1 2 8. Can't concentrate, can't pay attention for long

0 1 2 9. Can't get his/her mind off certain thoughts; obsessions (describe):

0 1 2 10. Can't sit still, restless, or hyperactive

0 1 2 11. Clings to adults or too dependent

0 1 2 12. Complains of loneliness

0 1 2 13. Confused or seems to be in a fog

0 1 2 14. Cries a lot

0 1 2 15. Cruel to animals

0 1 2 16. Cruelty, bullying, or meanness to others

0 1 2 17. Daydreams or gets lost in his/her thoughts

0 1 2 18. Deliberately harms self or attempts suicide

0 1 2 19. Demands a lot of attention

0 1 2 20. Destroys his/her own things

0 1 2 21. Destroys things belonging to his/her family or others

0 1 2 22. Disobedient at home

0 1 2 23. Disobedient at school

0 1 2 24. Doesn't eat well

0 1 2 25. Doesn't get along with other kids

0 1 2 26. Doesn't seem to feel guilty after misbehaving

0 1 2 27. Easily jealous

0 1 2 28. Breaks rules at home, school, or elsewhere

0 1 2 29. Fears certain animals, situations, or places, other than school (describe): _____

0 1 2 30. Fears going to school

0 1 2 31. Fears he/she might think or do something bad

0 1 2 32. Feels he/she has to be perfect

0 1 2 33. Feels or complains that no one loves him/ her

0 1 2 34. Feels others are out to get him/her

0 1 2 35. Feels worthless or inferior

0 1 2 36. Gets hurt a lot, accident-prone

0 1 2 37. Gets in many fights

0 1 2 38. Gets teased a lot

0 1 2 39. Hangs around with others who get in trouble

0 1 2 40. Hears sound or voices that aren't there (describe):

0 1 2 41. Impulsive or acts without thinking

0 1 2 42. Would rather be alone than with others

0 1 2 43. Lying or cheating

0 1 2 44. Bites fingernails

0 1 2 45. Nervous, highstrung, or tense

0 1 2 46. Nervous movements or twitching (describe):

0 1 2 47. Nightmares

0 1 2 48. Not liked by other kids

0 1 2 49. Constipated, doesn't move bowels

0 1 2 50. Too fearful or anxious

0 1 2 51. Feels dizzy or lightheaded

0 1 2 52. Feels too guilty

0 1 2 53. Overeating

0 1 2 54. Overtired without good reason

0 1 2 55. Overweight

56. Physical problems without known medical cause:

0 1 2 a. Aches or pains (not stomach or headaches)

0 1 2 b. Headaches

0 1 2 c. Nausea, feels sick

0 1 2 d. Problems with eyes (not if corrected by glasses) (describe):

0 1 2 e. Rashes or other skin problems

0 1 2 f. Stomachaches

0 1 2 g. Vomiting, throwing up

0 1 2 h. Other (describe): _____

0 1 2 57. Physically attacks people

0 1 2 58. Picks nose, skin, or other parts of body (describe):

0 1 2 59. Plays with own sex parts in public

0 1 2 60. Plays with own sex parts too much

0 1 2 61. Poor school work

0 1 2 62. Poorly coordinated or clumsy

0 1 2 63. Prefers being with older kids

0 1 2 64. Prefers being with younger kids

0 1 2 65. Refuses to talk

0 1 2 66. Repeats certain acts over and over; compulsions (describe):

0 1 2 67. Runs away from home

0 1 2 68. Screams a lot

0 1 2 69. Secretive, keeps things to self

0 1 2 70. Sees things that aren't there (describe): _

0 1 2 71. Self-conscious or easily embarrassed

0 1 2 72. Sets fires

0 1 2 73. Sexual problems (describe): _____

0 1 2 74. Showing off or clowning

0 1 2 75. Too shy or timid

0 1 2 76. Sleeps less than most kids

0 1 2 77. Sleeps more than most kids during day and/or night describe):

0 1 2 78. Inattentive or easily distracted

0 1 2 79. Speech problem (describe): _____

0 1 2 80. Stares blankly

0 1 2 81. Steals at home

0 1 2 82. Steals outside the home

0 1 2 83. Stores up too many things he/she doesn't need (describe): _____

0 1 2 84. Strange behavior (describe): _____

0 1 2 85. Strange ideas (describe): _____

0 1 2 86. Stubborn, sullen, or irritable

0 1 2 87. Sudden changes in mood or feelings

0 1 2 88. Sulks a lot

0 1 2 89. Suspicious

0 1 2 90. Swearing or obscene language

0 1 2 91. Talks about killing self

0 1 2 92. Talks or walks in sleep (describe): _____

0 1 2 93. Talks too much

0 1 2 94. Teases a lot

0 1 2 95. Temper tantrums or hot temper

0 1 2 96. Thinks about sex too much

0 1 2 97. Threatens people

0 1 2 98. Thumb-sucking

0 1 2 99. Smokes, chews, or sniffs tobacco

0 1 2 100. Trouble sleeping (describe): _____

0 1 2 101. Truancy, skips school

0 1 2 102. Underactive, slow moving, or lacks energy

0 1 2 103. Unhappy, sad, or depressed

0 1 2 104. Unusually loud

0 1 2 105. Uses drugs for nonmedical purposes (don't include alcohol or tobacco) (describe):

0 1 2 106. Vandalism

0 1 2 107. Wets self during the day

0 1 2 108. Wets the bed

0 1 2 109. Whining

0 1 2 110. Wishes to be of opposite sex

0 1 2 111. Withdrawn, doesn't get involved with others

0 1 2 112. Worries

113. Please write in any problems your child has that were not listed above: 0 1 2 _____

The items comprising each behavioral domain are:

Withdrawn: 42, 65, 69, 75, 80, 88, 102, 103, and 111.

Somatic Complaints: 51, 54, 56a, 56b, 56c, 56d, 56e, 56f, 56g, and, if applicable, 56h.

Anxious / Depressed: 12, 14, 31, 32, 33, 34, 35, 45, 50, 52, 71, 89, 103, and 112.

Social Problems: 1, 11, 25, 38, 48, 55, 62, and 64.

Thought Problems: 9, 40, 66, 70, 80, 84, and 85.

Attention Problems: 1, 8, 10, 13, 17, 41, 45, 46, 61, 62, and 80.

Delinquent Behavior: 26, 39, 43, 63, 67, 72, 81, 82, 90, 96, 101, 105, and 106.

Aggressive Behavior: 3, 7, 16, 19, 20, 21, 22, 23, 27, 37, 57, 68, 74, 86, 87, 93, 94, 95, 97, and 104.

Additionally, the CBCL had three broad-band scores: Internalizing, Externalizing and Total Problems.

The Internalizing scale is comprised of items from the Withdrawn, Somatic Complaints and Anxious / Depressed scales.

The Externalizing scale is comprised of items from the Delinquent Behavior and
Aggressive Behavior domains.

The Total Problems scale is comprised of all items, except for items 2 and
4 (allergies and asthma).

APPENDIX – 5
PANESS - Revised

Lateral preference pattern - "Show me how you"

1. EYE: Look thro a paper	L	R
2. FOOT: Kick ball	L	R
Stamp out fire	L	R
3. HAND: Comb hair	L	R
Brush teeth	L	R
Cut with scissors	L	R
Throw ball	L	R
Hit ball with bat	L	R
Hit ball with racket	L	R
Hammer	L	R

Use screw driver	L	R	
Saw	L	R	
Flip coin	L	R	MIXED: if <7R or L
(open door with key)			

Code no. of times child misses line or puts foot down flat out of 10 steps

	Side		Overflow		No. of errors			
4. Walks on heels					0	1	2	3
a. Hand posture present	L	R	0	1				
5. Walk on tiptoe					0	1	2	3
a. Hand postures present	L	R	0	1				
6. Walks on sides of feet					0	1	2	3
a. Hand postures present	L	R	0	1				
7. Tandem walk (heel to toe forward)					0	1	2	3
8. Tandem walk backwards					0	1	2	3

Code period of uninterrupted success for 20 seconds

9. Sustentations Postures / Stations	Overflow		Seconds			
	0	1	20	19-15	14-10	9-0
Put feet heel to toe, close your eyes and stand straight until I tell u to stop			0	1	2	3
a. Tendency to fall		0	1			
b. Arms out of balance		0	1			

10. Sustentation / Steadiness.	Overflow		Seconds			
	0	1	20	19-15	14-10	9-0
Put feet heel to toe, close your eyes, Raise your arms out in front of you, Spread your fingers apart and stay that way until I tell you to stop			0	1	2	3
a. Occurrence of involuntary movt.		0	1			
b. Tendency to fall		0	1			

Code 0 – No problem 1 – misses nose, wobbles en route

11. Finger to nose

a. Before you open your eyes, touch your R index finger to your nose 0 1

b. touch your L index finger to your nose 0 1

12. Stick out your tongue until I tell you to stop Seconds: 20 19-15 14-10 9-0

a. Occurences of involuntary movt. or lapses 0 1

(only score reptile tongue not curling)

Allow choice of first foot. Code choice for 13 &14

	Side		Seconds			
			20	19-15	14-10	9-0
13. Stand on one foot until I tell you to stop	L	R	0	1	2	3
Now stand on other foot	L	R	0	1	2	3

Code no. of hops until child touches down

14. Hop on one foot	L	R	0	1	2	3
Hop on the other foot	L	R	0	1	2	3

15-21 indicate time to do 20 taps. Circle side chosen first

Time		Proximal	Orofacial	Mirror	Overflow	Dysrhythmia	Slow	Unable
15. Foot tap	L:	0 1	0 1	0 1	0 1	0 1	0 1	0 1
	R:	0 1	0 1	0 1	0 1	0 1	0 1	0 1
16. Foot heel	L:	0 1	0 1	0 1	0 1	0 1	0 1	0 1
toe tap	R:	0 1	0 1	0 1	0 1	0 1	0 1	0 1
17. Hand pat	L:	0 1	0 1	0 1	0 1	0 1	0 1	0 1
	R:	0 1	0 1	0 1	0 1	0 1	0 1	0 1
18. Hand	L:	0 1	0 1	0 1	0 1	0 1	0 1	0 1
Pron/Sup	R:	0 1	0 1	0 1	0 1	0 1	0 1	0 1
19. Finger tap	L:	0 1	0 1	0 1	0 1	0 1	0 1	0 1
	R:	0 1	0 1	0 1	0 1	0 1	0 1	0 1
20. Fin. Succ	L:	0 1	0 1	0 1	0 1	0 1	0 1	0 1

R: 0 1 0 1 0 1 0 1 0 1 0 1 0 1

Indicate time to do 10 sets	Time	Overflow	Slow for age
21. Tongue wiggles 10 sets side to side	___		(if 3 secs)
a. Jaw synkinesia		0 1	1

Total Score:

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ABBREVIATIONS

NSS	-	Neurological Soft Signs
CBCL	-	Child Behaviour Check List
PANESS	-	Physical and Neurological Examination for Subtle Signs
HR	-	High Risk
SCHIZ	-	Schizophrenia
AFF. D	-	Affective Disorder

APPENDIX 1 - CONSENT FORM

ஒப்புதல் படிவம்

என் பட்டமேற்படிப்பு ஆராய்ச்சியின் ஒரு பகுதியாக உங்கள் குழந்தையை பரிசோதிக்கவும். உங்கள் பங்கேற்பிற்கும் அனுமதி கோருகிறேன்.

1. இதில் பங்குபெறுவது முழுவதும் உங்களின் சொந்த விருப்பத்திற்குரியது.
2. இதற்கு மறுப்பதன் மூலமோ, இதிலிருந்து விலகுவதன் மூலமோ உங்கள் கணவருக்கோ / மனைவிக்கோ கிடைக்கவேண்டிய மருத்துவ சிகிச்சை முறையில் எந்த குறைபாடோ, மாறுதலோ இருக்காது.
3. உங்களைப் பற்றிய சுய தகவல்கள் ஆராய்ச்சிக்கு மட்டுமே உபயோகப்படுத்தப்படும் என்று உறுதி கூறுகிறேன்.

என் ஆராய்ச்சி என்னவெனில்

மனநலம் பாதிக்கப்பட்ட பெற்றோரின் (தாய் (அ) தந்தை) குழந்தைகளிடம், ஏதேனும் மனநோய்குறிகள் (அ) நரம்பியல் அறிகுறிகள் இருக்கிறதா என கண்டறிவதே ஆகும். இதனை, ஒரு கேள்விப்பட்டியலின்

மூலம் உங்களிடமும், ஒரு எளிய, சிறிய நரம்பியல் பரிசோதனையின் மூலம் உங்கள் குழந்தையிடமும் செய்ய இருக்கிறேன்.

உறுதிமொழி

நீங்கள் மேற்கூறிய தகவல்களை படித்தேன். எனக்கு ஏற்பட்ட சந்தேகங்களை உங்களிடம் தெளிவுபடுத்திக் கொண்டேன். இந்த ஆராய்ச்சிக்கு என் பங்களிப்பையும் என் குழந்தையை பரிசோதிக்க அனுமதியையும் முழு மனதுடன் அளிக்கிறேன்.

நாள்: பெற்றோர்:

மருத்துவர்: குழந்தை:

MASTER CHART KEY

Consang – Consanguinity

DevDelay – Developmental delay

Birth compl – Birth complications

F/H – Family History

School perf – Scholastic performance

PMP – Premorbid personality

RxResp – Treatment Response

Ext – Externalizing problems

Child Diag – Diagnosis of the child

Int – Internalizing problems

NSS-L – Lateralization 1- Right, 2 – Left, 3-Mixed, 4-Change in Eyedness

DYSR-T – Dysrhythmia

Imper - Impersistence

Invol – Involuntary movement

Rpt-T – Repetitive movements Total

Ov-Excess – Overflow Excess	Ov-Asy – Overflow asymmetry
With – Withdrawn	Aggr – Aggression
Seq-T – Sequencing movements-Total	Soc – Social problems
Thot – Thought problems	Atten – Attention problems
Delin – Delinquency	Anx / Depr – Anxiety / Depression

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CERTIFICATE

This is to certify that the dissertation entitled a **"STUDY OF NEUROLOGICAL SOFT SIGNS AND PSYCHOPATHOLOGY IN CHILDREN OF PATIENTS WITH SCHIZOPHRENIA AND AFFECTIVE DISORDER"** is a bonafide record of work done by

Dr.PREETI. K., in the Department of Psychiatry, Government Rajaji Hospital, Madurai Medical College, Madurai, under the direct guidance of me.

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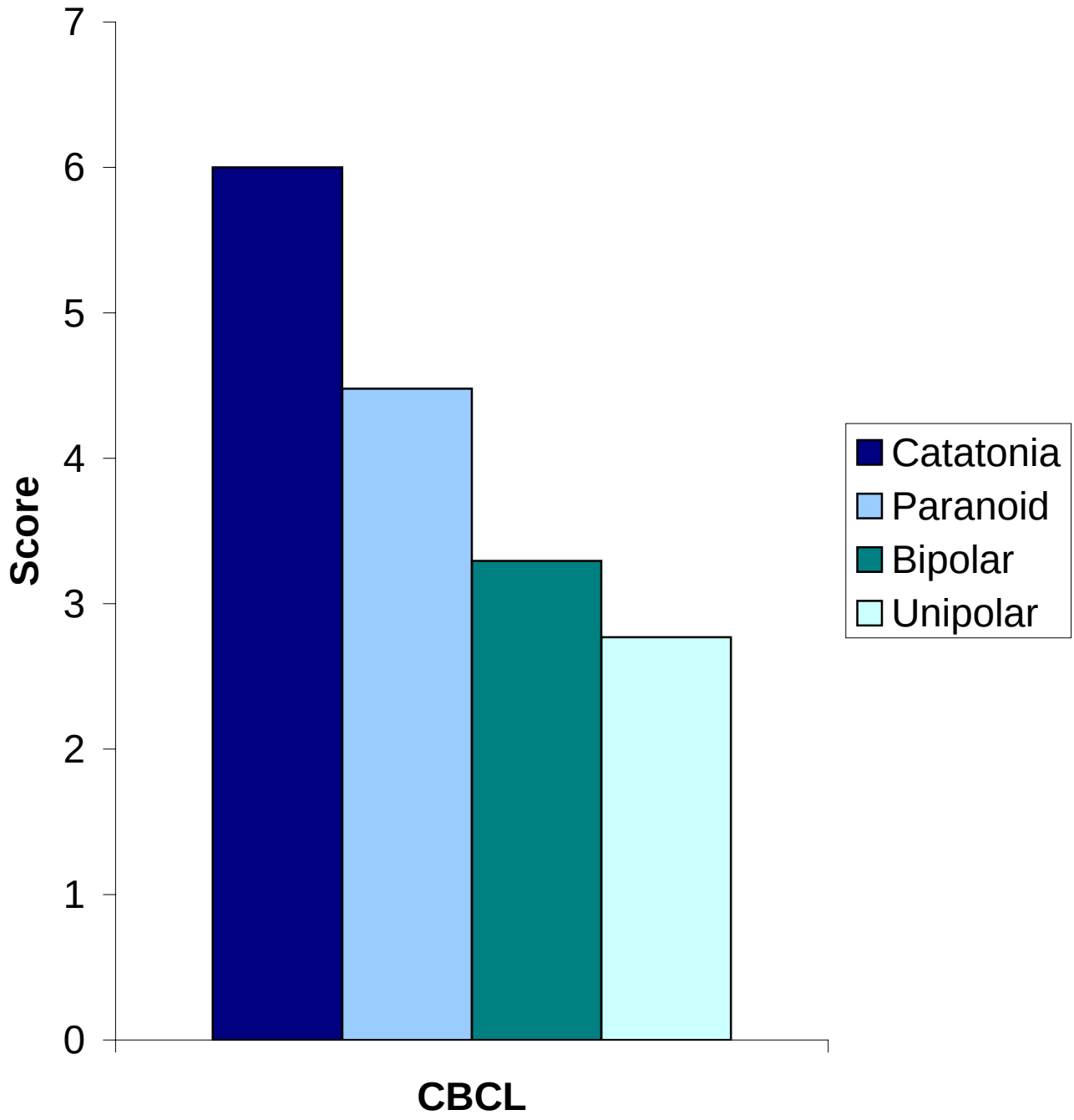
Madurai.

Fig 2. Comparison of CBCL Scores of the Children of the 4 Diagnostic Subgroups



Master Chart- Affective Dirsorder group

S.No	Child Age	Child Sex	Domicile	Consang	Birthcomp	DevlDelay	schoolperf	ill-Parent	Diagnosis	Age-Onset	Duration	F/H
1	8	M	Urban	Absent	Absent	Normal	Good	F	D	28	<2 years	Present
2	7	M	Rural	Absent	Absent	Normal	Good	F	D	44	<2 years	Absent
3	7	M	Urban	Present	Absent	Normal	Good	F	D	32	<2 years	Absent
4	14	M	Urban	Absent	Absent	Normal	Good	F	D	32	<2 years	Absent
5	9	M	Rural	Absent	Absent	Normal	Poor	M	BP	18	>2 years	Present
6	15	M	Rural	Present	Absent	Normal	Poor	M	BP	24	>2 years	Present
7	9	F	Urban	Absent	Absent	Normal	Good	F	D	25	<2 years	Absent
8	13	M	Rural	Absent	Absent	Normal	Good	F	D	25	>2 years	Absent
9	15	M	Rural	Absent	Absent	Normal	Poor	M	BP	47	>2 years	Present
10	14	F	Urban	Absent	Absent	Normal	Poor	M	BP	40	<2 years	Present
11	13	F	Urban	Absent	Absent	Normal	Poor	M	BP	40	<2 years	Present
12	5	F	Rural	Present	Absent	Normal	Good	F	BP	28	<2 years	Absent
13	8	M	Urban	Absent	Absent	Normal	Good	F	BP	29	>2 years	Absent
14	5	M	Rural	Present	Absent	Normal	Good	M	BP	26	>2 years	Absent
15	11	F	Rural	Absent	Absent	Normal	Good	F	BP	26	>2 years	Absent
16	10	F	Rural	Absent	Absent	Normal	Poor	F	BP	26	>2 years	Absent
17	9	M	Rural	Absent	Present	Normal	Good	F	BP	26	>2 years	Absent
18	14	M	Rural	Present	Absent	Normal	Poor	F	D	36	<2 years	Absent
19	15	M	Rural	Present	Absent	Normal	Good	F	D	36	<2 years	Absent
20	7	F	Urban	Present	Absent	Normal	Good	M	D	37	>2 years	Absent
21	12	F	Urban	Present	Absent	Normal	Good	M	D	37	>2 years	Absent
22	11	F	Urban	Absent	Absent	Normal	Good	F	BP	28	>2 years	Absent
23	13	M	Urban	Absent	Present	Delayed	Poor	F	D	22	<2 years	Absent
24	10	F	Rural	Absent	Absent	Normal	Poor	F	D	24	>2 years	Absent
25	8	M	Urban	Present	Absent	Normal	Good	M	D	25	>2 years	Absent
26	9	F	Rural	Present	Absent	Normal	Poor	F	BP	20	>2 years	Absent
27	8	M	Rural	Present	Absent	Normal	Poor	F	BP	20	>2 years	Absent
28	6	F	Rural	Absent	Absent	Normal	Good	F	BP	21	>2 years	Absent
29	9	M	Rural	Present	Absent	Normal	Good	F	BP	18	>2 years	Absent
30	12	M	Rural	Absent	Absent	Normal	Poor	M	BP	40	<2 years	Absent

PMP	Rx Resp	Burden	Child-Diag	NSS-L	GAIT	DYSR-T	IMPER	INVOL	RPT-T	Seq-T	Ov-Excess	Ov-Asy	NSS-Total	With
Introvert	Good	High	Nil	1	7	1	5	0	11	5	8	0	37	0
Extravert	Good	Less	Nil	1	8	1	1	1	10	5	1	2	27	0
Extravert	Good	Less	Nil	1	0	3	0	0	9	8	2	-3	22	0
Extravert	Good	High	Nil	4	2	3	0	0	12	1		0	18	0
Extravert	Good	High	Nil	1	6	2	4	0	8	5	4	2	29	0
Extravert	Good	High	Nil	1	0	3	8	0	10	1	3	-1	25	0
Extravert	Good	Less	Nil	1		2	0	0	10	10	4	-1	26	0
Extravert	Good	Less	Nil	1	3	1	0	0	5	1	1	0	11	0
Extravert	Good	Less	Nil	4	0	3	0	0	8	6	2	0	19	0
Extravert	Good	Less	Nil	1	0	2	0	0	1	0	0	0	3	0
Extravert	Good	Less	Nil	1	0	1	0	0	2	4	0	0	7	0
Extravert	Good	Less	Nil	1	2	1	0	0	1	2	0	-1	6	0
Extravert	Good	Less	Nil	1	3	1	3	0	9	2	6	0	24	0
Extravert	Good	Less	Nil	1	4	2	0	0	5	4	0	0	15	0
Extravert	Good	Less	Nil	1	3	1	0	0	2	0	0	0	6	0
Extravert	Good	Less	Nil	1	3	1	3	0	3	1	4	0	15	0
Extravert	Good	Less	Nil	1	7	0	4	0	7	2	5	0	25	0
Introvert	Good	High	Nil	1	1	0	0	0	6	2	1	1	10	1
Introvert	Good	High	Nil	1	0	1	0	0	6	0	0	0	7	0
Extravert	Good	Less	Nil	1	5	4	0	0	0	2	0	0	11	0
Extravert	Good	Less	Nil	1	0	2	0	0	0	0	0	0	2	0
Extravert	Good	Less	Nil	1	5	2	0	0	6	7	1	0	21	1
Introvert	Good	Less	Nil	1	3	8	0	0	7	9	3	-1	30	0
Extravert	Good	Less	Nil	1	7	S	0	0	4	5	6	-1	22	0
Extravert	Good	Less	Nil	4	5	0	0	0	6	3	0	1	14	0
Extravert	Good	Less	Nil	1	10	0	6	0	9	4	3	1	32	0
Extravert	Good	Less	Nil	1	12	4	6	0	9	8	1	1	40	0
Extravert	Good	Less	Enuresis	1	6	2	0	0	4	4	0	0	16	0
Extravert	Good	Less	Nil	1	5	2	0	0	9	3	4	-1	23	0
Extravert	Good	High	Nil	1	0	0	0	0	3	1	3	-1	7	0

Aggr	Soc	Thot	Attn	Delin	Anx/Dep	Somatic	others	CBCL-Total	Ext	Int
0	0	0	0	0	0	0	0	0	0	0
0	0	0	1	0	0	0	0	1	0	0
0	0	0	0	0	0	0	3	3	0	0
0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	2	0	0	0	2	2	0
2	0	0	2	0	0	0	2	6	2	0
0	0	0	0	0	0	0	0	0	0	0
4	0	0	2	0	0	0	0	6	4	0
0	0	0	0	0	0	0	2	2	0	0
0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	1	1	0	0
0	0	0	0	0	0	0	0	0	0	0
2	0	0	1	0	0	0	0	3	2	0
2	0	0	0	0	0	0	2	4	2	0
2	0	0	0	0	0	0	2	4	2	0
3	0	0	0	0	0	3	0	6	3	3
2	3	0	0	0	0	0	2	8	2	1
2	0	0	0	0	1	0	2	5	2	1
2	0	0	0	0	0	0	0	2	2	0
3	0	0	0	0	0	0	0	3	3	0
0	0	0	0	0	2	0	0	3	0	3
0	0	0	0	0	0	0	0	0	0	0
0	0	0	2	0	0	0	0	2	0	0
4	0	0	0	0	0	0	2	6	4	0
0	0	0	3	0	2	0	0	5	0	2
0	0	0	2	0	0	0	4	6	0	0
1	1	0	0	0	0	0	0	2	1	0
3	0	0	3	0	0	0	0	6	3	0
4	0	0	2	0	0	0	0	6	4	3